Vol. XLV. No. 169. AUTUMN, 1928.

The Bristol Medico-Chirurgical Journal

PUBLISHED UNDER THE AUSPICES OF

THE BRISTOL MEDICO-CHIRURGICAL SOCIETY.

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The Bristol Medico-Chirurgical Journal

" Scire est nescire, nisi id me
Scire alius sciret."

AUTUMN, 1928.

INDICATIONS FOR MANIPULATIVE SURGERY.*

BY

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[After an introduction in which the speaker gave his reasons for believing that manipulative treatment could and should be practised by medical men, particularly by surgeons who had acquired the necessary skill by practice, he went on to give some account of the principles indicating the advisability of using those plans of treatment.]

THE types of cases in which manipulation is indicated fall into five principal groups: (1) Cases with adhesions; (2) functional or hysterical cases; (3) unreduced dislocations and subluxations; (4) miscellaneous; (5) combined cases.

* Part of an address delivered to the Bristol Medico-Chirurgical Society at the University, on April 11th, 1928.

CASES WITH ADHESIONS.

Adhesions are bands of scar tissue due to organisation of inflammatory products which give rise to pain, limitation of movement, tenderness, weakness, and recurrent effusion. The most characteristic of these is a certain amount of limitation of movement. They occur frequently after injuries such as sprains, dislocations or fractures, and may be synovial, capsular or extra-capsular. They are very common also after diseases of joints, whether synovitis or arthritis. They are not always associated with joints, but may occur in connection with muscles, tendons and their sheaths, and connective-tissue planes. It cannot be too frequently emphasised that these adhesions are often, indeed usually, preventable by more enlightened treatment of injuries and diseases of joints and of fractures, *i.e.* by instituting early movement.

Examples of post-traumatic adhesions spring at once to mind—the stiffness of the shoulder following dislocation or fracture near the joint, the crippling stiffness of the wrist and fingers following a neglected Colles fracture, stiffness of the ankle and mid-tarsal joint after Pott's fracture, very many cases of flat foot with limitation of inversion, the persistent limitation of full flexion of the knee seen after many cases of sprain.

Why do we still see such large numbers of joints crippled by adhesions? It is largely due to the persistence of irrational and unphysiological methods of treating injuries and diseases of joints or of the locomotor apparatus generally.

The general treatment, with a view to the prevention of adhesions resulting from synovitis following a sprain or contusion of a joint, can be summed up in a few words, viz., "Rest combined with early movement through complete range." At first sight this maxim

may seem contradictory, but this in reality is not the case. Rest from weight-bearing and excessive use permit healing to proceed; but early movements, at first of but short duration, prevent the formation of adhesions. The principle of early movement, admirable though it be, is not sufficient, for unless we aim at early movement *through its full range* adhesions may yet develop and give rise to much subsequent trouble.

Let us attempt to illustrate the principle of the prevention of adhesions by a concrete example, viz. the treatment of a case of synovitis of the knee following a sprain of the internal lateral ligament.

Rest in bed or on a couch is enjoined at first. The next step consists in applying a firm compression bandage over a thick layer of wool, which should completely surround the joint and extend for some little way upwards into the thigh and downwards into the leg, leaving a small fringe of wool above and below uncovered by the bandage.

No splint should ever be used, but the joint is slightly flexed over a pillow, for experience has shown this to be the position of greatest ease. Rest from weight-bearing must be enjoined as long as the synovial effusion persists. From the very first gentle massage (effleurage), electrical stimulation of the quadriceps and movements may be instituted, for these, added to the elastic compression, play an important part in the absorption of the intra-articular effusion and prevent muscular wasting. Each day the movements are increased. In about a week in marked cases, or considerably less in minor cases, effusion will have subsided, and it should be our aim to have obtained full movements by this time.

There is now no objection to weight-bearing, provided strain upon the recently damaged ligament

be eliminated, and this can usually be ensured by the simple device of reinforcing the inner sides of the sole and heel by a wedge-shaped piece of leather $\frac{1}{4}$ in. in thickness. For a short period, and especially if there is any tendency to swelling after exercise, a compression bandage should be worn. This, however, should be dispensed with as soon as possible as it tends to interfere with the tone of the thigh muscles. As this swelling is often due to loss of tone of the thigh muscles, every effort should be made to remedy this by re-education.

Then, as examples of stiffness due to disease, we have the crippling disabilities following the so-called "rheumatoid arthritis," when the acuter symptoms have subsided. These cases are literally "crying out" for manipulation, and the risk of a flare-up is negligible if suitable cases are chosen and suitable technique employed. In many of these, of course, the ankylosis is of so firm a nature that manipulation is out of the question, although other surgical measures are full of promise. There is, however, a considerable percentage in which the stiffness is slight or moderate, but sufficient to cause considerable pain and disability. In many instances, by carefully restoring the normal range of movement we break down or loosen the adhesions and scar tissue that are the cause of the greater part of this pain and disability. The results of manipulation in suitable cases of this kind are remarkably good, especially if by suitable after-treatment the wasted muscles are made to recover their tone. I have performed manipulation on many hundreds of such cases, and have been greatly impressed by the possibilities of this method. Many of my patients have had every conceivable form of medical treatment, and this is, of course, logical during the more acute stages; but surely it is scarcely logical to attempt to

cure a joint stiffened by scar tissue by vaccines or drugs. The best results accrue in the cases in which the arthritis is or has been of the synovial or capsular type and the actual articular surfaces but little involved.

Adhesions may occur, not only in connection with joints but in muscles or in intermuscular planes, in connection with tendons and their sheaths, fasciæ, aponeuroses and ligaments. Local areas of scar tissue may occur after injuries in connection with these structures which may often be benefited greatly by manipulation. A good example of this lies in adhesions in the lumbar region of the back following an injury to the erector spinæ.

As another example the interesting condition known as "tennis elbow" may be chosen for discussion. That some cases of long standing that have defied every other form of treatment can be cured by manipulation is beyond doubt. The condition is usually attributed to the back hand stroke in tennis or racquets, but it occurs in persons who do not play these games. It is common, for instance, in hammermen and others who have to grip a thick handle tightly, and some hold that in tennis players it is due to playing with too thick a handle. Clinically there is pain and tenderness in the region of the external condyle of the humerus near the tendinous common origin of the extensor group, or over the posterior aspect of the radio-humeral joint, or both may be combined. There is weakness of grip, so that articles are dropped suddenly. If the fingers are flexed into the palm and the wrist then flexed, there is pain at the site first mentioned. According to Osgood, the symptoms are in many cases due to inflammation of a bursa in this region. I have never, personally, seen any evidence of such a bursa, but think that most cases

are due to a partial rupture of tendinous fibres with formation of minute adhesions, and that the cures following manipulation are due to rupture of these adhesions. In some cases periostitis without adhesions seems to be the most prominent feature, and in these manipulation is scarcely indicated.

[The lecturer then pointed out the dangers of manipulation in certain cases, particularly in tuberculous disease and in myositis ossificans.]

Myositis Ossificans. — As is well known, this condition is apt to follow an injury of, or in the vicinity of, a joint, particularly the elbow-joint in children, and there is a formation of new bone in muscle, ligament, tendon or other connective-tissue structures. The condition is probably due to the liberation of osteoblasts from beneath the torn periosteum, and it is characteristic that massage, passive movements and manipulation cause increased pain, swelling and muscular spasm, and appear to increase the rate of formation of new bone. There is no objection, however, if the joint has become fixed in a bad position, to manipulating it gently under anæsthesia into that position which experience has shown to be the best should ankylosis unfortunately occur.

FUNCTIONAL OR HYSTERICAL CASES.

It is in these cases that the most dramatic results are obtained by manipulation. Such cases not unnaturally attract a great deal of public attention; and when, as occasionally happens, the patient has been under the care of, not one, but many eminent surgeons, and the so-called miracle has been performed by a bone-setter, the medical men in question not infrequently come in for some severe criticism.

The diagnosis of a functional condition is often

extremely difficult, and depends to a great extent upon that indefinable asset intuition. This is a large and important subject, to which it is impossible to do justice in a short paper, and attention is merely drawn to a few special points which are sometimes overlooked.

1. An injury associated with marked mental shock, such as a railway or motor-car accident in which a joint is injured, is particularly liable to give rise to a neurosis in an individual of a nervous temperament. There is no surgeon of great experience who has not, at some time, been puzzled or misled by a functional condition of the joint.

2. A purely functional disturbance may occur from the first, or more frequently the condition is superadded upon some such basis as a slight degree of chronic synovitis or arthritis. The unwary or inexperienced may overlook the functional element, and consider that the symptoms and signs betoken a much more marked degree of arthritis than is actually present.

3. Quite apart from the question of injury, it must be remembered that a functional element is very frequently quite a marked feature in chronic arthritis in both sexes. These are the cases of "rheumatoid arthritis" of many years' standing which are cured by "faith-healers."

4. In an early functional case, if spasm be abolished by anæsthesia, full movement can be obtained readily. If, however, the condition is of long standing, actual adaptive shortening of ligaments and periarticular structures occurs. This is, of course, more liable to occur if the condition has been superimposed upon a slight degree of chronic synovitis or arthritis.

5. Even in primarily functional cases swelling of the joint may occur, probably from the general loss of tone of the thigh muscles and impaired absorption of

synovial fluid. It may then be a moot point whether the functional condition is primary or secondary.

6. Manipulation is by far the most effective therapeutic measure in these functional cases owing to the powerful suggestion of the anæsthetic. Treatment by suggestion should, however, precede and immediately follow the anæsthetic, and be carried on for some weeks or months subsequently.

7. The after-treatment is often of more importance than the manipulation itself, and not only the surgeon, but everyone with whom the patient comes in contact, must endeavour to convince the patient that he or she is now cured.

8. The prognosis in these cases depends upon the time that the condition has been present. In cases of long standing the prognosis is less hopeful. Even more, perhaps, depends upon the personality of the surgeon who performs the manipulation.

The type of case most frequently encountered is the chronic back, with which we are all probably familiar. There is usually a history of some injury, of moderate severity, such as a fall, thereafter there is a complaint of a dull ache in the back after any exercise, and the patient gradually becomes a semi-invalid, X-ray examination is negative, and other causes of pain in the back have been excluded. There is usually pain on stooping and recovering, and the back muscles are flabby. Very often these patients have either been told that there is nothing really wrong with the back or, on the other hand, have been led to believe that serious trouble is present. Not infrequently they have worn spinal jackets and supports over prolonged periods without relief. In this type of case the results of manipulation, followed by re-education of the spinal muscles, are often almost miraculous.

DISLOCATIONS AND SUBLUXATIONS.

This is another large group of cases in which manipulation is of the greatest value. The treatment of the ordinary dislocations by the classical methods is well known and need not detain us. A few words are necessary, however, concerning dislocations, or, to speak more accurately, "fracture-dislocations" of the semilunar cartilages. It has always been a profound mystery to me why this condition, so absorbingly interesting and so frequently encountered in practice, has been so neglected and left largely to bone-setters. It is extremely probable that a very large percentage of the patients of a well-known bone-setter recently retired, consisted of unrecognised cases of unreduced fracture-displacement of the semilunar cartilage. No little part of his reputation was due to his undoubted skill in these manipulations. It is regrettable that many of these patients had sought help from our profession in vain.

There is evidence that manipulation is not only of value in recent cases of fracture-dislocation of a semilunar cartilage, but also in some recurrent cases. A fracture-dislocation of a semilunar cartilage must be reduced immediately, and reduction has not occurred until the patient can fully and painlessly extend the knee. If extension is limited by only a few degrees, or if by an effort one can get almost full extension, but this is still painful, the cartilage is probably still unreduced. With appropriate treatment there should be no recurrence.

In the recent cases we must bear in mind that the displaced fragment, owing to its elasticity, has a pronounced tendency to spring back into position, provided the obstruction presented by the femoral condyle can be eliminated. I advise every one of my listeners who has not done so to take a femur and

tibia, articulate them, and settle for himself what are the manipulative movements that will most readily cause the displaced fragment to retrace its step. He will acquire a more accurate conception than by a perusal of the usually faulty text-book descriptions. The latter often advise a long period of immobility after reduction, and some even say that the limb should be put in plaster for a period. As a matter of fact, provided rotatory movements are avoided, the patient can start gentle exercises as soon as the acuter symptoms have subsided and effusion almost gone. Straightforward flexion and extension, combined with weight bearing, probably assist repair. Prolonged immobility leads to marked and sometimes irremediable wasting and stiffness. Permanent stiffness of a limb is not a cure for a torn cartilage.

In some chronic cases apparent cure occurs, but in some the benefit is of a temporary nature. This fact depends upon the physiology of repair in the semilunar cartilages. Repair is at best sluggish, and when the fractured surfaces have become smooth and endothelialised, as in cases of long standing, it is probable that actual repair occurs but rarely. How, then, can we explain the relief or even apparent cure that manipulation may bring about in these chronic cases?

A fact of fundamental importance in this connexion is that the commonest lesion of a semilunar cartilage is a definite tear which usually takes the form of a complete longitudinal tear or "bucket-handle" type of lesion, in other words it is a "fracture-dislocation." The characteristic symptoms of the semilunar derangement are due to the behaviour of the fractured portion of cartilage. It is most important to remember that in these recurrent cases it is quite possible for the inner torn portion to occupy a certain position without

giving rise to any " locking " or limitation of movement or, indeed, to any physical or clinical symptoms whatsoever. This position is the narrow space in the intercondylar region between the crucial ligament and the femoral condyle.

During rotation of the tibia upon the femur rotation takes place in the arc of a circle, the centre of which is at the tibial attachments of the crucial ligaments. It follows that during any sudden rotatory movement the movement in the interior of the joint near the centre of the circle (near the crucial ligaments) is necessarily less than at the periphery. It follows, also, that in a given case of chronic " bucket-handle " lesion of a semilunar cartilage, in which the surfaces have become smooth and rounded and actual repair probably impossible, the displaced fragment might be less likely to give rise to symptoms in the position aforesaid than if actually restored to its original position.

I have performed successful manipulations upon many of these recurrent cases of semilunar lesion with the avowed intention of driving the fractured portion into the interior of the joint. In my opinion a successful result depends upon converting an incomplete longitudinal tear into the complete variety, and the manipulative technique differs appreciably from that necessary for recent cases.

It may be argued that at operation in these recurrent cases the displaced fragment is usually found in the interior of the joint, where it obviously gave rise to symptoms. However, in most of these cases the tear is not absolutely complete, but is such that a certain degree of tension is present, sufficient to cause recurrent displacements.

To sum up, the commonest lesion of a semilunar cartilage is a definite tear, and most commonly the

complete longitudinal tear or "bucket-handle" type. At the original accident the inner torn portion is usually dislocated into the interior of the joint, where, being fixed at first between the femoral and tibial condyles, it gives rise to a mechanical block to extension.

By manipulation two things may happen: (1) In more recent cases the displaced portion may retrace its steps and, provided rotary movements are avoided for some time, repair may take place—in more chronic cases a trial may be given to the same method; and (2) if it fails the displaced portion may be forced still farther into the interior of the joint, where, in many cases, it may give rise to no further trouble for considerable periods, although recurrences often occur.

Manipulation in recurrent displacements of the semilunar cartilages, although well worth trying, is not infallible, particularly in professional footballers and others whose work is strenuous. It is well worthy of trial in persons whose work or play is not of a strenuous nature. The results of operative removal of the torn cartilages are so satisfactory in experienced hands that this should be usually advised when there is a history of several recurrences. The patient should be up and walking fairly well ten to fourteen days after the operation, and although one occasionally hears a contrary opinion expressed, experience teaches that removal of the cartilage does not prevent participation in the more strenuous forms of work or sport.

Within the short space at my disposal it has obviously been only possible to touch briefly upon a few aspects of treatment by manipulation. I trust, however, that sufficient has been said to indicate the very great possibilities of affording relief or of curing disability that lie in this neglected field, to all who believe that Dogma is the enemy of Progress.

THE COLLOIDAL STATE OF MATTER.

BY

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IN 1861 Graham found that substances which dissolved in water could be classified into two groups. Bodies like sugar, salt and urea in solution rapidly diffuse through parchment, and these solutions deposit crystals on evaporation; this group was termed Crystalloids. On the other hand, glue, dextrin, gelatin in solution either diffuse very slowly or not at all, and as a general rule such solutions do not crystallise on evaporation; this group was termed Colloids. It is preferable to speak of a substance as being in the "colloidal state" rather than allude to it as a "colloid," for in all probability all substances can be caused to assume this state.

In true solutions the dissolved substance is either in the molecular or ionic condition, and such solutions are looked upon as homogeneous mixtures; colloidal solutions, however, are heterogeneous. These latter solutions may be clear and transparent to the naked eye, and no microscope—used in the normal way—reveals the solid phase, but if a beam of light is passed through them, the beam viewed at right angles to its path, the track of that beam appears turbid; in true solutions the track is invisible. The light diffracted is often polarised, indicating that the particles must have dimensions comparable to the wave length of the illuminating beam. If this beam is examined

with a microscope placed at right angles to the track—*i.e.* the "ultra-microscope"—the diffraction images of the separate particles are rendered visible. The particles themselves are invisible; it is the light reflected from them only that is seen. If the liquid is optically clear (a pure solvent, or solutions of crystalloids) the field of view is dark; if matter is present in the colloidal state, minute points of light are seen against the dark background. These are in more or less rapid motion. If a colloidal solution is being examined, the field of view appears to be filled with brilliantly-lit particles; "like a swarm of dancing gnats in a sunbeam, they spring, hop, dance and fly apart so rapidly that the eye can hardly follow them."

The diameter of a blood corpuscle is about one eight-thousandth of a millimetre, a "micron" μ = ·001 mm., hence a corpuscle is about 8 μ. Most micrococci are about 1 μ. Particles of colloidal gold are about seven to fifteen-millionths of a millimetre; as 1 $\mu\mu$ = ·000001 mm., such colloidal material lies between 7 and 15 $\mu\mu$. A molecule of chloroform is about 0·8 $\mu\mu$, and a molecule of hydrogen 0·1 $\mu\mu$.

The characteristic properties of material in the colloidal state are largely due to the immense development of surface. A small lead shot converted into this state develops a surface many million times that of lead in the form of shot.

The motion of matter in the colloidal state is due to the ceaseless movement of the molecules in the interior part of all liquids. The particles behave as if they were true gas molecules with kinetic energy.

The colloidal particles are electrically charged, and many of their properties, especially their stability, depend on the nature of this charge. Silver chloride, or lead selenide, for instance, are negatively charged,

and the solution bathing them positively. On the other hand, lead prepared by Bredig's method, or ferric hydrate, is positively, and the liquid in which they exist negatively, charged.

Substances in the colloidal condition can be divided into two groups. (1) *Suspensoids* (or suspensions or sols), such, for instance, as lead, silver, selenium, iodine, lead selenide, silver bromide; all are either positively or negatively charged in solution in water, and are precipitated on discharging, negatively charged colloids by positively charged ions, or positively charged particles by negatively charged ions. During this discharging process the precipitated colloid carries with it a part of the precipitating ion, a phenomenon known as adsorption and highly characteristic of colloidal phenomena. (2) *Emulsoids* (or jellies or gels), such as gelatin, gum arabic, glue, starch, dextrin, etc., etc. These also carry electrical charges, are heavily hydrated, but are not precipitated by dilute solutions of electrolytes. Heat, however, or the addition of concentrated solution of salt or other solvents, acting upon the hydrated colloidal particles, effects coagulation in such cases.

The difference between suspensoids and emulsoids is a matter of degree; probably the latter occupy a position intermediate between suspensoids and true solutions. Under the ultra-microscope emulsoids show a general diffuse light. There is not in these solutions the same optical contrast between the two phases as there is in the case of suspensoids. Emulsoids, particularly those not sensitive to precipitation by electrolytes, increase to a surprising extent the stability of suspensoids towards such electrically charged ions. Consequently, colloidal solutions of lead selenide, for instance, may be stabilised by the addition of solutions

of gum arabic; in such cases the latter substance is termed the *protective colloid.*

Within recent years an ever-increasing number of substances has been obtained in the colloidal state by methods which cannot be described here. Suspensoids of various elements, such as lead, selenium, iodine, etc., and compounds such as lead selenide, etc., have been used in medicine, but biologically emulsoids are by far the more important group. The latter contain starch, glycogen, proteins, lecithin, enzymes, etc., etc., and even their enumeration is sufficient to show that all the phenomena of life are connected with processes that take place only in colloidal systems.

REDUNDANT COLON: A GROUP OF CASES EXHIBITING SYMPTOMS TRACEABLE TO THIS CONDITION.

BY

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THE normal adult colon has an average length of 2 metres, and if longer than this cannot fit into the body without forming extra loops, reduplications or even kinks and angulations. Being mobile, these loops need not be fixed or constant in position, nor need they give rise to any symptoms *provided the colon is healthy and functioning normally and regularly.*

With regard to their etiology, Kantor,[1] who has studied nearly 700 cases, considers that they are congenital, and even hereditary and familial in their transmission. But it is probable that this is by no means the whole truth, and that in many cases the condition is acquired.

From 9 to 14 per cent. of all people show these redundancies, and the incidence among constipated people is as high as 23 per cent. (Kantor). They are not generally associated with visceroptosis, being commoner in the "sthenic," active, strong type of person.

All cases show a predisposition to imperfect colonic function, and 77 per cent. of Kantor's cases had

long-standing constipation; the greater the looping the greater the constipation.

Other symptoms which can often be traced to the presence of these redundancies, when associated with impeded function, are: (1) *vague abdominal discomfort* (all cases in this series); (2) *irregular pain*, often of a colicky nature due to commonly associated spasm (Cases 2, 3, 4, 7 and 8); (3) *gas distress* (all cases); (4) *precordial pain and palpitation* following gas-collection under the left leaf of the diaphragm (Cases 3 and 4); (5) *belching or hiccough* and epigastric pain with discomfort after meals due to the same cause as (4) and to the misdirected efforts of the patient to rid himself of gas that seems to be in the stomach (Cases 4 and 6); (6) *mucous stools* (Cases 2, 4 and 6), occasionally with blood (Case 6); (7) *alternating constipation and diarrhœa* (Cases 2, 4 and 7); (8) *vomiting* (Case 7); (9) *mental depression, carcinophobia, etc.* (Cases 4, 6 and 7); (10) *rectal symptoms* from portal congestion and hæmorrhoids (Cases 5, 6 and 7).

These symptoms are not due to the mere presence of the redundancy, but to associated spasm, atony and stasis, and to the fermentation, excessive gas-formation and toxic absorption resulting from the latter.

A redundancy in the distal part of the colon may produce symptoms localised in the proximal half, suggestive of appendicitis or of biliary colic, due to a "throw-back" of the fæcal column and retention.

The diagnosis is suggested by (*a*) a long history of constipation (this was not present in Case 8); (*b*) a long interval between stools; (*c*) occasional enormous evacuations. It can be positively confirmed by an opaque-meal X-ray examination, followed by an opaque enema for preference.

Before discussing the treatment of the condition there are a few points that need clearing up in connection with its pathology. Are such redundant loops in any way causal or contributory to the symptoms so often found associated with them, or are they rather effects or negligible by-products of colon mal-function in a congenitally long colon? In a healthy colon a "loop" is not a fixed or static condition, but obviously if it become overfilled, displaced or distorted it can impede the forward motion of the fæcal column by (1) kinking and by (2) interference with local blood supply, producing irregular neuro-muscular action and abnormal mucosal function. The latter shows itself in a gradual loss of normal neuro-muscular propelling power, a tendency to spasm and colic, and a deficient absorption of CO_2 and CH_4 into the blood stream, thus augmenting the commonest symptom, viz. gas-distress. Note the presence of gas distal to the loops in all the cases illustrated (except Case 1 and Case 8, who had an opaque enema). Moreover, if the state of the musculature of the loop become so atrophic that the loop becomes practically a passive tube, then we have the condition of a regular plumber's trap, a local cesspool, the contents of which include a residue that is changed less and less and tends to increase.

Once colon mal-function begins, then, in a person having a congenitally long colon, the redundant loop becomes highly important as a link in the chain of cause and effect, and a vicious circle is very easily induced.

Treatment.—Surgical measures are contra-indicated except in the rare cases where volvulus has occurred or where definite evidence of adhesions or marked obstruction is found. (The mobility of the loop can be

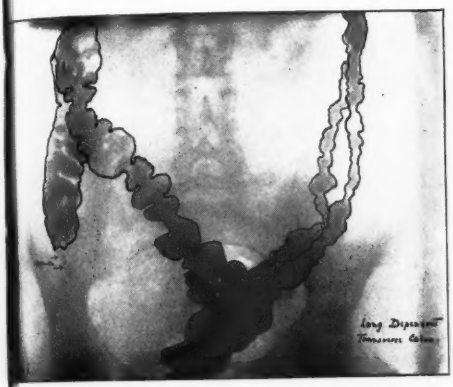
tested under the X-rays by systematic palpation, and its emptying rate observed.)

In the attempt to restore normal colonic function the presence of the redundant loops is a complication which was most satisfactorily met in the majority of this series of cases by judicious abdominal massage and exercises, the masseur regulating his manipulations by constant reference to a skiagram showing the exact anatomy of the colon in question. Cathartic and enema-habits should be suspended, a rough bulky diet and plenty of water should be prescribed, together with lubrication by oil taken by mouth after meals and, if it can be retained, *per rectum* at night (6 oz.), in the early stages. If spasticity is present, increasing doses of belladonna are valuable (10 to 25 minims t.d.s.).

In these cases the very fact of relieving the patient's mind by assuring him of the absence of serious disease is a powerful factor in successful treatment. The condition can be explained to him, and he should be told that in his case it is not necessary for the bowels to move every day. In Case 6 the effect of this line of treatment was dramatic: after a month's treatment he was a totally different man. Intelligent patients can be shown by the masseur how to massage their own abdomens and what exercises to perform, so that the need of prolonged special treatment is dispensed with.

Relapses may occur, and every effort should be made to prevent the patient from becoming introspective, and to encourage him to take regular exercise and to interest himself in things outside his own ego. The vicious circle can thus be broken and normal colonic function restored, but it must always be remembered that the presence of such an abnormality

PLATE IV.



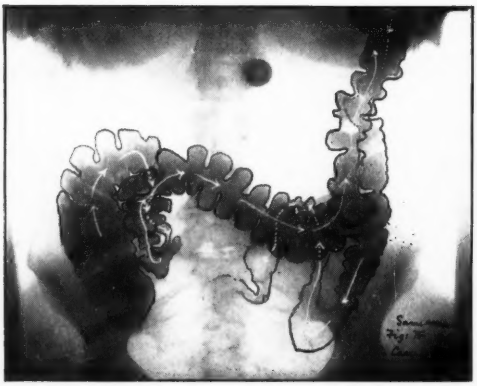
CASE 1.

CASE 2.

CASE 3.

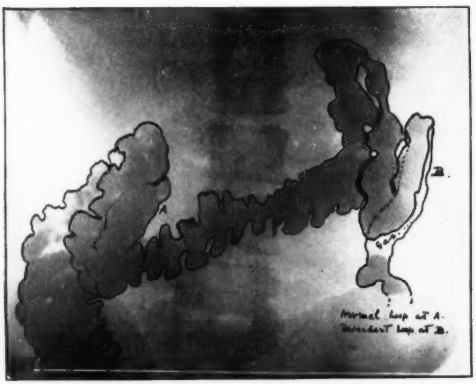
CASE 4.

PLATE V.

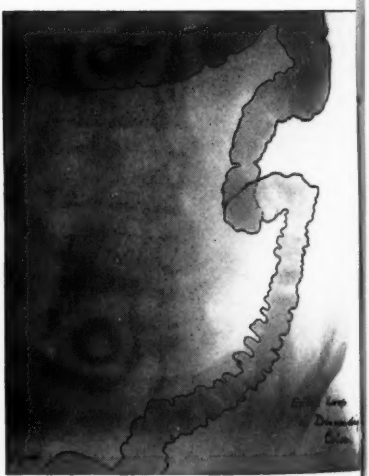


CASE 4.

CASE 6.



CASE 7.



CASE 8.

in the colon constitutes a permanent disability and increases the liability to relapse.

Notes on the Cases and Illustrations. (*All Opaque Meals except Case* 8.)

In the appended illustrations the colon has been outlined in Indian ink solely in order to show more clearly, in the reproductions, the *main direction* taken by the loops, some of them lying in several different planes only recognisable on screen examination at different angles.

Cases 1 and 2.—Long dependent transverse colons with no loops, but in Case 2 causing symptoms. Case 2 had coloptosis of proximal half, " splenic drag " and gas-distress.

Case 3.—A complication of the condition seen in Case 1. Angulated loop in transverse colon. Proximal retention, spasm and distal gas-distress.

Case 4.—(Figs. 4 and 5.)—Proximal retention, marked gas-distress, colic.

Case 6.—Marked degree of redundancy. " Fulness " and gas-distress, no pain, but intense depression, loss of weight and lack of interest in life.

Case 7.—Indigestion for years (though stomach and duodenum found to be normal). Hiccough. Pain in left hypochondrium. Constipated. Occasional vomiting.

Case 8.—(Enema.)—Recurrent attacks of pain, sometimes severe, under left costal margin. Very active man, always healthy, bowels regular. No vomiting or nausea.

SUMMARY.

1. The anatomy, etiology and incidence of redundant colon are described.

2. Symptoms traceable to this condition in a mal-functioning colon as observed in the cases selected are detailed.

3. The question as to how far the condition can be considered pathological is discussed.

4. The treatment is outlined.

5. Notes and illustrations of seven selected cases are given.

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THE LYMPHOCYTOSIS OF INFECTION.*

BY

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DURING the past fifteen years, and especially during the last seven years, considerable interest has centred round the cases which have been variously described under the title of "the lymphocytosis of infection," "infectious mononucleosis," "acute benign lymphoblastosis," and "glandular fever," which though not very common are yet sufficiently frequent to be of importance, both from the theoretical and practical standpoint. I have therefore ventured to bring forward the following case, which was of great interest to me.

Mrs. R. V., æt. 30, residing on the South Coast, had nursed her children with pertussis, and had contracted a bad cough herself, which was possibly pertussis also, for it lasted from April 19th through May, 1927, after which she had been quite well till she went to a ball on July 7th, where she partook of a pasty of some form of meat which she thought must have "turned," for it tasted nasty. She did not, however, have either diarrhœa or vomiting subsequently, and on the next day, July 8th, she bathed in the sea, a pastime of which she was very fond.

On July 9th she was taken with severe headache, which was quite unusual to her, and the headache continued subsequently for a fortnight, with a temperature continuing between 100° and 102°.

* A paper read before the Bath and Bristol Branch of the British Medical Association, April 25th, 1928.

On July 23rd she travelled to Bath, and when I saw her that evening for the first time the auxiliary temperature was 101·4°, and her face was flushed, with a heavy expression strongly suggestive of typhoid. The spleen was enlarged, and could be felt one to two inches below the costal margin, but there were neither spots nor diarrhœa. The liver border was also palpable. All the cervical glands, in the anterior and posterior triangles, including the supraclavicular regions, were enlarged, as also the right axillary and to a lesser extent the left axillary glands. The gums bled at the margins on touching, which she noticed herself when brushing the teeth.

On the morning of the next day, July 24th, I took blood films, the differential count of which showed polymorphs 11–13 per cent. only, lymphocytes 85–89 per cent., eosinophils 4 per cent. A general blood count made the same afternoon showed red blood corpuscles 4,600,000 per c.mm., leucocytes 14,000, so that there was a considerable relative increase, and a smaller absolute increase of lymphocytes.

On July 25th Dr. Odery Symes kindly saw her with me, when blood was taken for agglutination tests against *B. typhosus* and *Paratyphosus A and B*, and it was felt that, if these proved negative, the possibility of acute leukæmia could not be excluded without further watching. The agglutination tests proved negative. Examination of ocular fundi showed neither hemorrhages nor other serious ophthalmoscopic change, nor was any evidence of tubercle found there, or in the lungs, though it may be noted that her sister had died of tubercular meningitis (acute tuberculosis) in infancy. One other point of interest in the history is that I had attended Mrs. V. as a child for a typical attack of glandular fever, but at that time the blood examinations, which would have made the records more complete, were not usually made in such cases. The urine contained a trace of albumen.

That evening, while thinking somewhat anxiously over this case, I recollected having seen a similar case recorded by Dr. A. J. Hall,[1] under the title of "A Case Resembling Acute Lymphatic Leukæmia, Ending in Recovery," and from the details there given we were encouraged to hope that our patient's illness also might come to a favourable issue. I will refer to Dr. Hall's case later.

We thought it well to try and exclude any septic focus, and though a dentist on the South Coast had recently pronounced favourably upon the teeth, I asked Mr. Buchan to see Mrs. V. with me, as there was an upper canine with a

sinus near its apex, and one or two lower roots to be seen. Mr. Buchan found that five teeth were dead, and advised their extraction, a few at a time to avoid risk of hemorrhage or severe reaction. After preliminary administration of calcium lactate, extractions were performed on July 29th, August 5th, 15th and September 14th, the first three under gas, the last with gas and ether, a skiagram being finally taken to identify any further septic teeth. The canine, which was root filled, had the largest apical abscess attached to the root which I remember to have seen. All the extracted teeth were unhealthy and several had granulations at the apex. A definite reaction with rise of temperature followed the extraction of the right lower molar, but otherwise Mrs. V. made steady progress towards recovery symptomatically, though a blood film taken on August 1st still showed the percentage of lymphocytes to be as high as 85 per cent.

On September 3rd the total number of leucocytes in the film taken was decidedly smaller, with polymorphonuclears 28 per cent., lymphocytes 67 per cent. Only the tip of the spleen could now be felt on deep palpation, and the glands in the neck were practically normal. The patient looked wonderfully better, and was now just beginning to walk in the garden. On October 5th, 1927, the red blood corpuscles were fully normal or over; hæmoglobin 75 per cent.; leucocytes 4,000 per cmm., with polymorphonuclear average 49 per cent., lymphocytes 49·45 per cent. The spleen could no longer be felt, and she returned to her home on the South Coast, and I did not see her again till April 17th, 1928, when she looked in excellent health, and the blood count averaged about 8,000 leucocytes with polymorphonuclears 55 per cent., lymphocytes 42 per cent. There was no enlargement of glands or spleen, and no tendency of the gums to bleed.

While the percentage of lymphocytes is still a trifle above the normal, I think there can be no doubt that this case is not one of leukæmia, but falls under the category of the "lymphocytosis of infection." Let us now look in a little more detail at the recorded cases of the lymphocytosis of infection, taking them chronologically. Osler, writing in 1907,[2] and Dawson Williams in 1907[3] and 1909[4], on Glandular Fever, while describing clearly its symptomatology, make no

reference to examination of the blood. The first allusion to this is by some observers in Queensland in 1915,[5] who mention a lymphocytosis occurring, in some cases accompanied by a rash, which possibly may not be of the same nature as those to which we refer.

Dr. A. J. Hall's case, previously mentioned, was that of a gentleman taken ill in March, 1911, with a temperature of 101°, which was followed a few days later by an exudate covering the right tonsil, with enlargement of glands in the right and left sides of the neck, axillæ and groins, enlargement of the spleen and slight albuminuria. The blood examination showed red blood cells 5,400,000, leucocytes 32,000, with polymorphonuclears only 4 per cent., and lymphocytes 96 per cent. Gradual recovery took place, and he was able to go shooting in August, 1911. In 1914 the blood showed still a lymphocytosis of 46·6 per cent.

At the discussion on this case Osler thought that the onset was certainly very suggestive of acute lymphatic leukæmia. Parkes Weber suggested that it was a case of the "lymphocytosis of infection" described by Cabot,[6] and Dr. Hall agreed. Dr. Hall also found a paper by Marchand[7] and concludes: "Both Cabot and Marchand allude to the difficulty of diagnosing these . . . cases from early acute leukæmia until their rapid recovery makes it obvious, and therefore I gather that other observers have fallen into the same pit in times past."

We may now refer to Cabot's and Marchand's papers in abstract:—

Cabot's Cases.

1. A post-mortem wound, followed by lymphangitis, adenitis, continued fever, lymphocytosis not over 70 per cent., leucocytes not over 20,000. Slow but complete recovery.

2. A case of boils in an undergraduate, with persistent lymphocytosis of 82 per cent. Total leucocytes 3,400. Recovery.

3. A girl, æt. 20, during a raging epidemic of streptococcal sore throat developed sore throat. Temperature 102°. The cervical glands, lateral and posterior, were the size of marbles, in the right axilla the glands were the size of a hen's egg, the left axillary glands smaller ; spleen and liver were not palpable. There was dullness at each pulmonary apex and over the upper sternal region. Leucocytes 9,000, polymorphonuclears 28 per cent., lymphocytes 71 per cent. Recovery followed.

4. A man æt. 37 stated that he had " a cold " in February, 1912. On March 9th, while at the barber's, he had an attack of vertigo. A troublesome cough continued, and on March 18th there was enlargement of glands in the left side of the neck, the soreness of which passed off in three days. There was slight fever for ten days accompanied by night sweats. The blood count on March 28th showed a leucocytosis of 30,500, with polymorphonuclears 25 per cent. and lymphocytes 75 per cent. On May 18th there was a leucocyte count of 18,800, with polymorphonuclears 57 per cent. and lymphocytes 42 per cent. Recovery followed.

Marchand's Cases.

1. Male, æt. 50, typical paratyphoid fever. Polymorphonuclears 43 per cent., lymphocytes 55 per cent.

2. A case of lobar pneumonia, March, 1911. Readmitted November, 1911, with enlarged glands in the neck and at the angles of the jaw. The spleen formed a hard tumour. X-ray examination showed that the thorax was normal. Leucocytes 7,000, lymphocytes 33 per cent. Wassermann test and examination for tubercle bacilli were both negative. The enlarged glands and spleen were still palpable in January, 1912.

4. V. M., æt. 14. This case might now be regarded as one of glandular fever with hemorrhagic nephritis.

5. J. K., November, 1910, æt. 20, was admitted for fever, albuminuria, retinal hemorrhages, enlarged glands in neck, axillæ and groins, slight bronchitis, enlargement of left side of heart with systolic murmur. Pulse 120 to 130, typhoidal state. Blood examination : red corpuscles 1,500,000 per c.mm., hæmoglobin 35 per cent., leucocytes 2,100 per c.mm., lymphocytes 85·5 per cent., polymorphs 6·5 per cent. Death occurred December 12th, 1910.

The diagnosis at first was typhoid, at the end leukæmia or pseudo-leukæmia ; finally, the post-mortem showed streptococcal invasion with punctiform hemorrhages of brain and lungs, and streptococcal foci without suppuration.

Leaving the record of American epidemics recorded by Longcope and others, two papers by H. L. Tidy, the first in association with Morley in 1921 and the second in conjunction with Daniel in 1923, sum up the state of our knowledge of glandular fever, or infectious mononucleosis, while giving an account of an epidemic in a school of thirty boys, of whom twenty-four contracted the disease.

They define it as an acute infectious and contagious disease, principally of but not confined to childhood, characterized by marked enlargement of the cervical glands, and by a less constant and marked enlargement of the spleen, axillary, inguinal and other glands. There may be some tenderness of the cervical glands, but they are not acutely painful: abdominal pain is occasionally severe. The fauces are often reddened, there may be an enlarged tonsil, but exudation is unusual, and changes are slight compared with the marked glandular enlargement. There is no anæmia in the acute stage, but some malaise and enlarged glands by the third day. There may be pyrexia, about 103°, but it may be as high as 105° for a day or two, rarely over 100° for over a week. The glandular enlargement subsides in five to fifteen days ; it may relapse, or, if on one side, pass to the other. Suppuration is extremely rare. The glands may remain palpable for weeks or months, then a prolonged period of depressed health and anæmia follows, before recovery is finally complete.

Hemorrhagic nephritis occurs in 6 per cent. of the cases. The condition has no relationship to mumps,

whooping cough, measles, scarlet fever, tonsillitis or to leukæmia, Hodgkin's disease, tuberculosis or pyogenic infections. The incubation is normally from seven to ten days. Epistaxis may occur. The blood examination shows a lymphocytosis which may take time to develop and be transient only.

Tidy goes on to a discussion, and asks the following questions :—

1. "Is glandular fever a clinical entity?" To which he answers affirmatively.

2. "Are glandular fever and infectious mononucleosis identical?" To which he replies : "Clinically indistinguishable."

3. Tidy denies that septic infections have any connection with glandular fever. My case appears, however, to show that a connection between the two may exist. The cases of Cabot and Marchand also point definitely to an infection as a causal element, such as a post-mortem wound, a persistent furunculosis, a streptococcal throat, an attack of paratyphoid, or a fatal attack of septicæmia, to which may be added J. Fitch Landon's[10] cases.

Case 1.—A white, unmarried woman, æt. 26, was attacked with a mild pharyngitis in February, 1924, with a temperature of 99·4° on the fourth day of illness, pains and prostration. There were two tender glands below the left angle of the jaw, about the size of peas, and a few shot-like, painless, inguinal glands, but no enlargement of liver or spleen. Landon was called again on the fourteenth day as she had not improved, and found the cervical glands a little larger and more tender and the spleen palpable. To exclude malaria a blood examination was made which showed leucocytes 12,000 per c.mm., of which 86 per cent. were mononuclears, the majority being abnormal, somewhat larger than large lymphocytes, with pale blue cytoplasm and oval lobulated or kidney-shaped nuclei which stained intensely.

On the nineteenth day an eminent medical consultant considered the case to be one of acute lymphatic leukæmia. The throat got much worse, and on the twenty-second day there was typical Vincent's angina, which was confirmed by laboratory examination. There was increase in the size and number of the glands of both sides of the neck, and also of the axilla. Two applications of arsphenamin to the throat were followed by rapid improvement, and on May 9th, 1924, mononuclears had fallen to 58 per cent., and in February, 1925, to 28 per cent., only 3 per cent. being abnormal.

Case 2.—A coloured girl, æt. 16 years, admitted May 21st, 1920, complaining of pain in the upper abdomen for eight months. She had had follicular tonsillitis for two days. The urine contained a trace of albumen but no casts. Throat swabs were negative to diphtheria, and both Widal and Wassermann tests were negative, as were also the results of rectal and vaginal examinations. There were no tubercle bacilli in the sputum, but X-ray examination of the chest showed a large node at the hilus of the left lung. Blood examination: leucocytes 9,400 per c.mm., mononuclears 80 to 97 per cent., mostly large with "horseshoe" nuclei, no cytoplasmic granules. Persistent increasing temperature with loss of flesh and strength was followed by death five weeks after admission. Three members of the staff concurred in the diagnosis of acute lymphatic leukæmia. The autopsy showed tuberculous bronchopneumonia of upper lobe of left lung, tuberculous tracheal and bronchial lymphatic glands, and miliary tubercle in liver and spleen. The bone marrow was normal.

Landon comments on these cases, that "many non-leukæmic conditions with distinct lymphocytosis may occur, rarely however so marked as to suggest leukæmia," mentioning in children congenital syphilis, scurvy and rickets and pertussis, and in adults tuberculosis, malaria, hæmophilia, typhoid, pellagra. It may occur after the administration of thyroid extract, tuberculin, arsphenamin and pilocarpin, in tetrachlormethane poisoning, and (as mentioned above) Cabot's and Marchand's cases of streptococcal infection.

Tidy and Daniel[9] state in their paper: "In agreement with Longcope, Sprunt and Evans, and others, we consider that the cells are lymphoid in origin. . . . We regard the cells as slightly immature lymphocytes." Fitch Landon says in his paper[10] that "the cytoplasm of these mononuclear cells is greater in volume, and stains more deeply, than that of normal small lymphocytes. Their nuclei are irregular and frequently notched. Degenerating forms are absent in contrast to early acute lymphatic leukæmia. With oxidase stain these cells do not show granules indicating marrow origin, and hence are believed to be of lymphoid origin."

In the case of my patient, Mrs. R. V., the cells were mostly indistinguishable from lymphocytes, chiefly of the small variety, staining deeply with Leishman's stain, but having a greater number showing notching or indentation of the nucleus than in normal blood. Re-examined on October 6th, 1928, her blood showed over five million red corpuscles per c.mm., and 4,000 leucocytes, 52 per cent. of which were mononuclear cells whose reaction to the oxidase method proved them to be lymphocytes.

SUMMARY.

A case of continued fever, with enlargement of the lymphatic glands and spleen, and associated with a high lymphocyte count, is described. Recovery ensued after radical treatment of dental sepsis.

Similar cases from the literature are reviewed, and the relation between the lymphocytosis of infection and that of acute lymphatic leukæmia is discussed.

[Since I wrote this paper Dr. Odery Symes has kindly drawn my attention to a statement in the American periodical, *Medicine* (February, 1928, p. 8), to the effect that in over 50 per cent. of cases of dental apical sepsis a lymphocytosis occurs.]

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THE VALUE OF THE CHLORIDE CONTENT OF THE CEREBRO-SPINAL FLUID IN DIAGNOSIS AND PROGNOSIS.

BY

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THIS short paper is based upon examinations of cerebro-spinal fluids withdrawn by lumbar puncture from a series of 22 cases. Its object is to bring to notice the value of the estimation of chlorides in the cerebro-spinal fluid in both the diagnosis and prognosis of various conditions included in the general term "meningitis."

Since 1912, when Mestrezat[1] published his monograph on normal and pathological cerebro-spinal fluids, the value of cytological and chemical investigations has become firmly established. The literature upon this subject is considerable, but references to the chloride estimation in particular are few. During the last few years the most valuable contributions to our knowledge of this subject have been the work of Dr. J. G. Greenfield, at the National Hospital for the Paralysed and Epileptic, and I shall quote freely from these.

Willcox and Lyttle,[2] writing on the chemical composition of the spinal fluid in 1923, came to the conclusion that "the chlorides are likely to vary

directly with the acuteness of the infective process, and may bear some constant relation to the disease itself." Greenfield and Carmichael[3] are more emphatic, and state that "the chlorides in cerebro-spinal fluid are so constantly between 0·72 per cent. and 0·75 per cent. that any variation above or below these amounts is always of pathological significance."

The fluids obtained from five of the cases in this series appeared to be normal in all respects, and therefore serve as "controls." The percentages of chlorides in these cases were 0·73, 0·72, 0·74, 0·72 and 0·75 respectively, giving an average of 0·73 per cent. These figures agree with the normal limit laid down by Greenfield in the statement quoted above. A rise in the chloride percentage above 0·75 is said to be indicative of renal inadequacy, but I have no case of this type in which renal efficiency tests were carried out. The majority of the remainder of the cases show a fall in the chloride percentage, which is associated, in a general way, with meningitis.

The cause of the fall in chlorides is still unknown, but most investigators attempt to explain it as being due to a change in the composition of the blood, to a change in the permeability of the choroid plexus, to bacterial activity, or to a combination of these phenomena.

Greenfield's[4] view seems to me to be the most acceptable at the present time, *i.e.* that "this fall in the chlorides is due to a breaking down of the barrier which separates the blood and cerebro-spinal fluid. As Mestrezat termed it, there is an increase of meningeal permeability. In health the chlorides in the cerebro-spinal fluid are always higher than in the blood, but when the barrier between the two fluids is reduced their chloride percentages tend to approximate to one another."

In the consideration of the value of the chloride estimation for diagnostic purposes in meningeal infections it must be remembered that a fall in the chloride percentage in the cerebro-spinal fluid occurs in certain other conditions, such as lobar pneumonia, typhoid and typhus fevers, and in tuberculosis. It is evident, therefore, that this estimation must always be used in conjunction with other investigations of the fluid, for instance, the cytology, protein estimation, globulin, etc. This does not in any way detract from the value of the estimation, but rather enhances it; for it is the modifying influence of a knowledge of the chloride percentage on the picture given by the other investigations that makes it of such importance.

A knowledge of the chloride percentage is of special value in cases of meningitis secondary to inflammatory or suppurative processes elsewhere in the body, particularly in the middle ear, mastoid or other cranial air sinuses. For instance, a case of chronic middle ear suppuration showing clinical signs of meningitis, with increased cells and protein in the cerebro-spinal fluid, may have a widespread meningitis or may not. A chloride percentage within normal limits in such a case would indicate that the meningeal involvement was still localised, and that a timely operation to deal with the source of infection might avert the serious catastrophe of generalised meningitis. Greenfield,[4] reviewing a series of seventeen cases of head injuries, says, in connection with chloride estimations, that "this means that the chlorides always fall when meningitis becomes generalised, and therefore so long as they remain high there is ground for belief that the area of inflammation is localised and for hoping that it may remain so."

Seven cases of this series fall into this group of "secondary meningitis."

The first, with a chloride percentage of 0·585, was a fatal case of widespread meningitis secondary to otitis media.

The second, a boy 9 years of age, had meningitis secondary to mastoiditis. The chloride percentage in the cerebro-spinal fluid was 0·63. Operation relieved the condition and he went out cured.

The third case was one of frontal abscess secondary to sinusitis, with a chloride of 0·685 per cent. and a cell count of 88 polynuclears per c.mm., indicative of an established meningitis.

The fourth case was one of an old cerebellar abscess which began to leak, setting up a generalised meningitis. The cell count was very high, 8,000 polymorphs per c.mm., and a chloride percentage of 0·625. An interesting feature of this case was that a pure culture of Friedlander's pneumobacilius was obtained from the abscess cavity.

The fifth case, presenting signs of cerebellar abscess and having a history of otorrhœa, showed a cell count of 500 per c.mm., polymorphs and lymphocytes in equal proportion, and a chloride percentage of 0·715. At operation an extradural abscess was found accounting for the findings in the cerebro-spinal fluid, but further exploration for the cerebellar abscess was unsuccessful.

The sixth case was admitted to hospital unconscious and hemiplegic. Three months previously the blood had given a triple-positive Wassermann reaction, and a diagnosis of cerebral hemorrhage was favoured. The cerebro-spinal fluid was yellowish in colour, showed increased protein and globulin, and a chloride percentage of 0·70. Post-mortem examination revealed an extradural abscess over the petrous portion of the temporal bone, and a small collection of pus on the surface of the cerebellum.

The seventh case was one of suppurative otitis media ; the cerebro-spinal fluid was greenish in colour, the cell count showed 3,600 polymorphs per c.mm. and a chloride of 0·69 per cent. Operation failed to relieve her, and at post-mortem examination there was extensive purulent meningitis due to pneumococcal infection from the ear.

The fifth and sixth cases appear to me to be examples of fairly localised conditions in which operation is indicated, the deciding feature in each being the relatively high chloride percentage in a cerebro-spinal fluid which is otherwise typical of generalised meningitis. Yerger,[5] writing in 1925 on the intracranial complications of nasal and aural conditions, depends entirely upon the cell count of the cerebro-spinal fluid for differentiation between circumscribed and diffuse meningitis. I have not been able to find any satisfactory basis for differentiation by this method; in fact, the cell count can be very misleading.

For example, I have mentioned one case of diffuse meningitis with a cell count of 88 polymorphs, and another case of localised abscess with a count of 500 polymorphs. Differentiation by means of the chloride estimation is more reliable, but tends to be too delicate. The initial involvement of the meninges in these cases may so rapidly pass on to a generalised meningitis that the fall in chlorides may be very sudden, and twenty-four hours will suffice to lower a percentage from normal to about 0·675. For this reason cerebro-spinal fluids from cases of secondary meningitis often show a chloride percentage of about 0·7 or 0·69, and in spite of immediate operation the next sample shows a much lower percentage. But there is at least a chance that the condition will subside after the removal of the cause.

Of the remaining ten cases in this series five are of tuberculous meningitis, four of meningococcal meningitis and one of post-vaccinal encephalitis. In the cases of tuberculous meningitis the chloride percentages were 0·64, 0·628, 0·68, 0·63 and 0·665 respectively. Greenfield regards chloride percentages between 0·6 and 0·5 as pathognomonic of tuberculous

meningitis, and indeed it is customary to associate this condition with "lymphocytes and low chlorides."

For this reason there is a tendency to forget that a meningitis in a child, with a cell count chiefly polynuclear and only a moderate reduction of chlorides, may also be of tuberculous origin.

The case which showed a chloride percentage of 0·64 gave a cell count of 43 per cmm. mainly polynuclear, and the case showing a chloride of 0·665 per cent. gave a cell count of 400 with polynuclears and lymphocytes in equal proportion. This last case was also interesting in that the patient was twenty years of age. At no time during her illness could Kernig's sign be elicited. Tubercle bacilli were present in the cerebro-spinal fluid during life, and the diagnosis was also verified post-mortem.

In the four meningococcal meningitis cases the chlorides were 0·615, 0·585, 0·66, and 0·675 per cent. respectively on first examination. The first of these cases improved rapidly under treatment, and the chlorides rose steadily. The second case, showing the initial low chloride percentage of 0·585 improved to 0·64, then relapsed to 0·6; he again improved and chlorides reached 0·705 per cent. Another slight drop occurred to 0·695 per cent., and the case continued in a chronic condition. He eventually left hospital apparently cured. This relapse occurring in meningococcal meningitis at the end of the first week of treatment is of fairly common occurrence, and the reason generally given is that the serum is not reaching the ventricles in sufficient concentration to deal with the infection.

The variations in the chloride estimations which occurred in this case closely followed the actual clinical state of the patient, and I find that a record of the

chloride percentages in successive samples of fluid from a given case forms a most reliable index of progress. The last case of meningococcal meningitis remained undiagnosed for some days, owing to failure to isolate the organism, and during that time the chlorides fell from 0·675 to 0·595 per cent. When the exact nature of the infection became evident serum treatment produced a rise in the chloride percentage to 0·665, but the patient died the following day. Caffey and McLean,[6] reviewing thirty cases of meningococcal meningitis, conclude that the chloride estimations of the cerebro-spinal fluid do not give diagnostic or prognostic information of great value, but it seems that they regard the normal range of chlorides as 0·725 to 0·675 per cent., whereas any percentage below 0·70 is considered pathological in this country.

The last case of the series is that of a child admitted to hospital unconscious, ten days after vaccination. The condition was diagnosed as being a post-vaccinal encephalitis. The cerebro-spinal fluid showed 0·2 per cent. total protein, no increase of globulin, a cell count of 16 per cmm. chiefly polynuclears, and a chloride percentage of 0·74. She left hospital apparently cured.

The number of cases of secondary meningitis dealt with in this paper is quite insufficient to give conclusive evidence of the value of the chloride estimation in the differentiation between circumscribed and diffuse meningitis, although they indicate that such a possibility does exist. This estimation is, therefore, worth further investigation, and a greater amount of consideration is due to it than it has received in the past. As an index of progress a record of the chloride percentage in successive samples of fluid from any given case of meningitis is of definite value in

prognosis ; the upward curve of percentages encourages a hopeful outlook, while falling percentages are of bad omen. I have shown, however, that the chloride percentage may fall to a very low level, and recovery eventually ensue. From this it follows that in established meningitis no single chloride estimation is of any value in prognosis.

Greenfield,[4] however, says that he has never known a patient die from meningitis with the chlorides at their normal level.

Case No.	Age.	Clinical Features.	Cerebro-Spinal Fluid.			
			Cells per c.mm.	Protein per cent.	Globulin.	Chlorides per cent.
1	—	Normal.	2	0·02	No increase.	0·73
2	47	Normal.	1	0·015	No increase.	0·72
3	—	Normal.	3	0·015	No increase.	0·74
4	30	Normal.	2	0·015	No increase.	0·72
5	47	Normal.	3	0·015	No increase.	0·75
6	39	Meningitis secondary to otitis media. Confirmed by autopsy.	p.	0·2++	Increased.	0·585
7	9	Meningitis secondary to mastoiditis. Operation followed by recovery.	725 p.	0·15	Increased.	0·63
8	15	Meningitis. Frontal abscess, secondary to sinusitis. Confirmed by autopsy.	88 p.	0·025	No increase.	0·685
9	16	Meningitis. Old cerebellar abscess found post-mortem.	8,000 p.	0·05	—	0·625
10	40	Meningitis. Extradural abscess, secondary to otitis media. Confirmed at autopsy.	500 equal p. and l.	0·03	No increase.	0·715

p = polymorphonuclears. l = lymphocytes

Case No.	Age.	Clinical Features.	CEREBRO-SPINAL FLUID.			
			Cells per c.mm.	Protein per cent.	Globulin.	Chlorides per cent.
11	42	Cerebral hemorrhage suspected during life. Extradural abscess, with localised suppuration on cerebellar surface, found post-mortem.	—	0·2	Increased.	0·7
12	61	Meningitis secondary to otitis media. Confirmed by autopsy.	3,500 cells, 75 per cent. p.	0·2	Increased.	0·69
13	$1\frac{a}{12}$	Tuberculous meningitis. Bacilli in C.S.F. Fatal.	43 p.	0·045	Increased.	0·64
14	$4\frac{a}{12}$	Fatal. No autopsy.	Many l.	—	—	0·628
15	—	Fatal. No autopsy.	112 l.	0·045	—	0·68
16	$1\frac{a}{12}$	Bacilli in C.S.F. Fatal.	73·50 per cent. p.	0·15	—	0·63
17	22	Bacilli in C.S.F. Diagnosis confirmed at autopsy.	400, p. and l. equal.	0·1	Increased.	0·64
18	6	Meningococcal meningitis.	Many p.	0·025	—	0·615
19	12	Meningococcal meningitis.	Many p.	0·2++	Increased.	0·585
20	$\frac{a}{12}$	Meningococcal meningitis.	Many p.	0·05	—	0·66
21	43	Meningococcal meningitis confirmed at autopsy.	Many p.	0·05	No increase.	0·675
22	7	Post-vaccinal encephalitis. Recovery.	16 p.	0·02	No increase.	0·74

p=polymorphonuclears. l=lymphocytes.

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MENINGOCOCCAL SEPTICÆMIA.

WITH REPORT OF A CASE.

BY

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ALTHOUGH the case to be described presents no new points, the rarity of the disease in this country, which is more frequently recognised in America and on the Continent, makes it worthy of record.

CASE REPORT.

History of Present Illness.—On December 17th, A. B., male, aged 12, was admitted to the Bristol General Hospital, with the history that five weeks previously he was suddenly taken ill with a "chill." This was accompanied by a succession of crops of small red papules distributed on the face, limbs, mostly on the extensor surfaces, and on the abdomen. These faded in about four days, some leaving a faint stain. Three weeks ago he began to have pains in his knees and arms. For the last three weeks his temperature had been irregular. (It was not taken regularly before this time.)

Previous History.—"Consumptive bowels" as a baby. Mumps and measles several years ago.

Family History.—Father and mother alive and healthy. Five other children healthy. No miscarriages.

Condition on Examination.—Patient rather pale. Appetite good, sleeps well, bowels regular, micturition normal. Teeth good; tongue furred.

Generalised red maculo-papular eruption. In one or two places drying pustules, possibly due to scratching.

Heart not enlarged. Faint systolic bruit all over præcordium. No abnormal physical signs in lungs.

Abdomen normal in appearance. Spleen just palpable, below left costal margin. Liver not palpable.

No head retraction or Kernig's sign. Knee-jerks present and plantar reflex flexor. Pupils react to light and accommodation. Optic discs and fundi normal.

Urine: specific gravity 1016, reaction acid. No albumin, blood or sugar.

Subsequent Progress.—December 19th, 1927. Rash most marked in febrile periods; thought by dermatologist to resemble a drug rash. Patient quite comfortable in afebrile periods. Treated with salicylates.

December 29th, 1927.—Condition unchanged. Hexamine being given by mouth. Blood culture negative.

January 2nd, 1928.—Leucocytes 8,000 per c.mm.; polymorphonuclears 66 per cent. No malarial parasites seen in films.

January 4th, 1928.—Quinine given by mouth.

January 5th, 1928.—Blood culture negative. Blood shows no agglutination with Bacillus typhosus, paratyphosus A and B or with micrococcus melitensis.

January 6th, 1928.—Given 5 c.c. of 0·5 per cent. solution of mercurochrome intravenously.

January 9th, 1928.—Urine: no abnormality in deposit. Culture sterile.

January 16th, 1928.—Condition unchanged, given 0·15 gm. stabilarsan intravenously.

January 21st, 1928.—Patient vomited several times and complained of headache.

January 23rd, 1928.—Patient much worse. Lies on side with knees drawn up. Head retraction and Kernig's sign present. Lumbar puncture. Fluid under increased pressure and turbid. Laboratory report: "Protein much increased. Chlorides 0·585 per cent. Many polymorphonuclear leucocytes. Intracellular Gram-negative diplococci. Culture shows meningococci." Intrathecal anti-meningococcal serotherapy instituted.

January 27th, 1928.—Patient very ill. Has severe headache. Delusions at times.

February 22nd, 1928.—General condition improved. Cerebro-spinal fluid report: Protein 0·1 per cent. Globulin increased. Chlorides 0·695 per cent. Cells 162 per c.mm., polymorphonuclears and lymphocytes. A few cocci still present. White cell count: 7,000 per c.mm.; polymorphonuclears, 58 per cent.

February 27th, 1928.—Optic discs show slight optic neuritis both sides. General condition better. Meningeal symptoms nearly clear, but fever and rash continue.

March 13th, 1928.—Cerebro-spinal fluid clear. Protein 0·15 per cent. Chlorides 0·695 per cent. Cells mainly mononuclears. No organisms seen. Culture sterile. Serum given intravenously, followed by severe reaction.

March 16th, 1928.—Serum given intravenously.

March 22nd, 1928.—No meningeal symptoms remain. Temperature normal. No rash.

March 31st, 1928.—Some pain on micturition. Urine: reaction acid. Deposit: large numbers of red blood cells, fair number of white cells. No casts.

April 12th, 1928.—Patient apparently quite well. Urine: deposit shows small number of pus and red blood cells. No casts. No tubercle bacilli. Culture sterile.

April 20th, 1928.—No abnormality in urine.

April 23rd, 1928.—Patient discharged well. When last seen, ten days after discharge, he remained well, and free from any evidence of organic disease. The cause of the hæmaturia was not solved; possibly it was due to the administration of serum.

SUMMARY OF LITERATURE.

The first record of the isolation of the meningococcus from the blood was made by Osler[1] in 1898 from a case of meningococcal meningitis. In 1902 Salomon[2] reported a case of primary meningococcal septicæmia with intermittent fever and a maculopapular eruption, which terminated in meningitis after two months and ultimately recovered.

In 1906 Andrewes[3] reported a case of meningococcal septicæmia, which proved fatal in four days, with no sign of meningeal involvement. In 1908 Liebermeister[4] described a case of septicæmia, due to the meningococcus, that recovered without meningitis. Since that date many cases of meningococcal septicæmia and of extrameningeal meningococcal infections have been reported. Cecil and Soper[5] record a case of meningococcal endocarditis, as do also Warfield and Walker[28] and Schottmüller.[29] Herrick[6] reports cases of meningococcal empyema and of bronchopneumonia,

both without meningitis. Findlay[7] records a case of polyarthritis in which meningococci were found post-mortem in the joints. Jacobitz[30] describes cases of pneumonia due to the meningococcus.

It is now generally considered that all cases of meningococcal meningitis begin with a short phase of bacteriæmia. Herrick,[8] in 315 cases of meningitis, was able to make the diagnosis before the onset of meningitis by means of blood cultures in 40 per cent. He found that the average length of this invasive stage was forty-eight hours. Blackfan[9] states that once the infection is localised in the meninges there is usually no bacteriæmia.

Herrick, writing in Osler and McCrae's *Modern Medicine*,[21] divides a meningococcal infection into three stages :—

1. A stage of coryza, tonsillitis and pharyngitis. This may not progress and the patient may become a chronic carrier.

2. An invasive stage of bacteriæmia, usually lasting forty-eight hours, which may be prolonged for days or weeks.

3. A metastatic stage. In 90 per cent. the dominant metastasis is in the meninges, but it may occur in the joints, in the pericardium, endocardium, lungs or epididymis.

According to Goodall and Washbourn[22] septicæmic forms of meningococcal infection may be of three types :—

(*a*) Fulminating types, rapidly fatal with no localisation.

(*b*) Abortive types with rash and fever, lasting two or three days to a week and terminating in recovery, with no meningitis.

(*c*) Chronic types which persist for weeks or months

and may or may not be complicated by meningitis. Rolleston[23] agrees with this.

Boulanger-Pilet[10] describes seven types of pure septicæmia :—

1. A pseudo-malarial type with intermittent fever, an eruption usually maculopapular, but sometimes morbilliform, scarlatiniform or herpetiform, waxing and waning with the fever; transitory joint and muscle pains, and a polymorphonuclear leucocytosis.
2. A purpuric type which may be rapidly fatal.
3. A typhoid type.
4. An articular type with arthritis which may be purulent.
5. Mixtures of the above type, *e.g.* beginning as a typhoid type and becoming pseudo-malarial.
6. A type with abnormal termination. The infection may become localised in unusual situations, causing such lesions as pleurisy, endocarditis and iridocyclitis.
7. A "forme fruste" corresponding to Goodall and Washbourn's abortive type.

For these infections the French have coined the word "Meningococcémies."

Apart from the fulminating cases proving rapidly fatal in a few hours, often with a purpuric eruption (Herrick),[6] the usual course is that of the "type pseudo-palustre" of Boulanger-Pilet.[10] The main clinical features are an irregular, intermittent fever associated with a maculo-papular eruption and fleeting joint and muscle pains. In the apyrexial intervals the patient appears perfectly well (Herrick,[6] Neergard,[14] Bloedorn,[11] Vignot[12]). The rash is in some cases described as being like erythema nodosum (Liegois and Foulon[13]). There may be no rash (Vaucher and Schmid[15]) or no joint or muscle pains (Gandy and

Boulanger-Pilet[17], Gandy and Deguinard[19]); or merely irregular fever with headache and malaise (Netter,[16] Debré, Ravine and de Pfeffel[18]). The spleen is often palpable and there is a polymorphonuclear leucocytosis somewhere in the neighbourhood of 15,000 per c.mm. The case described is unusual in that at the times the blood was examined the highest number of leucocytes found was 8,000 per c.mm. A blood culture, unless made on special media, may be negative, as in the case reported (see also Bloedorn,[11] Neergard,[14] Gandy and Deguinard[19]). There may be slight transient albuminuria (Gandy and Boulanger-Pilet[17]); or, more rarely, a subacute nephritis leaving a persistent albuminuria (Gandy and Deguinard[19]). There may be a transient epididymo-orchitis.[19]

These cases usually terminate in a meningitis, and, as in the case described by Netter,[16] may last as long as 102 days. Less often they may terminate without localisation, a general septicæmia persisting throughout the whole course of the disease; or there may be localisation in some abnormal site causing iridocyclitis, pneumonia, endocarditis,[10] etc. More rarely this condition may supervene upon an apparently cured meningitis.[20] There may be relapses and recurrences of the meningitis.[19] In the pure septicæmias and before the onset of meningitis the cerebro-spinal fluid shows no changes ([17, 19, 15]).

The disease appears to occur more commonly in children. The diagnosis may be made, in the opinion of some authors,[19] from the intermittent fever, rash and joint pains, even in the presence of negative blood cultures. In the case of negative blood cultures confirmation of the diagnosis may be obtained by means of agglutination or complement deviation reactions ([16]). The differential diagnosis has to be made from malaria,

abortus fever, typhoid, and at first the condition may, as in this case, be diagnosed as acute rheumatism. The rash may easily be mistaken for a drug rash. Often the diagnosis remains uncertain, as in the case described, until the occurrence of meningitis.[14]

The pathogenesis is obscure, but such cases as those reported by Gandy and Deguinard,[19] in which a recrudescence of meningitis occurred, and that of Lauvergne and Carrot,[20] in which septicæmia followed an apparently cured meningitis and the exhibition of an autogenous vaccine, favour the view that the process is not a septicæmia in the generally accepted sense, but that continually repeated invasions of the circulation occur from some isolated focus in or near the meninges. There is, however, no proof as yet forthcoming of this view, which is therefore entirely speculative.

As regards treatment, the efficiency of different methods is debated. Many cases, as the present one, appear to have been cured by the exhibition of anti-meningococcal serum, either subcutaneously, intramuscularly or intravenously ([19] [11] [6] [18] [13] [31]). Others have found serotherapy absolutely ineffectual, and, of these, some record success with autogenous or stock vaccines in resistant cases. Courtois-Suffit and Garnier's case[26] terminated fatally after the administration of serum and vaccines had proved useless. Vaucher and Schmid[15] consider that their patient was cured by the injection of 10 c.c. of sterile milk, whereas Netter[16] found this inefficacious, as also uroformin, colloidal metals, quinine and novarsenobenzol. His patient was cured by the injection of sterile pus from a fixation abscess. Good effects following the production of a fixation abscess are recorded by other workers.[20]

Luton,[24] discussing the diminished value of serotherapy in meningococcal infections since the

war, attributes it to the increase in prevalence of meningococcus B (or type II. of Gordon), which is known to be intractable to serotherapy. He describes recovery from meningitis and from septicæmia following the use of a meningococcal endoprotein. At first he was inclined to regard the action of this as specific. He now considers it to be due to non-specific protein shock. Hermann and Lifschitz [25] record a case recovering spontaneously after the ineffective exhibition of quinine, iron and arsenic. This observation may be ranked with that of Monziols and Loiseleur, [27] who record the complete cure of a patient following the withdrawal of blood for a blood culture with no other treatment.

SUMMARY OF CASE.

The striking features of the case described were :—

1. The difficulty of diagnosis until the development of the meningitis. Acute rheumatism, malaria, "abortus" fever and miliary tubercle were among the diagnoses considered at different times.

2. At no time when the white cells were counted was there a marked leucocytosis.

3. The prolonged course of the pyrexia (see chart). This persisted for ten weeks before, and for another seven weeks after meningitis developed.

4. Intrathecal injection of anti-meningococcal serum was without effect on the pyrexia, although improving the meningitis, which ran a very protracted course. Both were promptly arrested after the intravenous and intramuscular injection of polyvalent anti-meningococcal serum.

I should like to express my gratitude to Dr. Rogers for the cerebro-spinal fluid reports, and to Dr. Carey Coombs, under whose care the boy was, for permission to publish and for much help and advice.

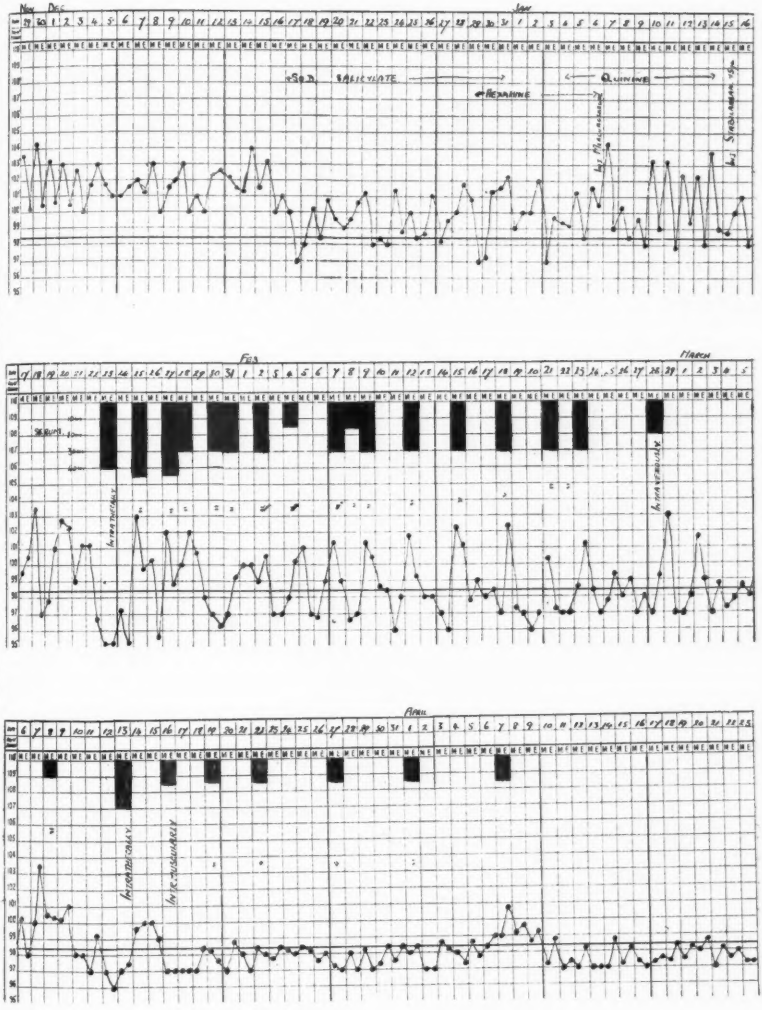

Chart showing the influence of Anti-Meningococcal Serum on the course of the disease.

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CHEMOTHERAPEUTIC RESEARCHES ON CANCER.*

A REVIEW.

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The recent International Conference seems to have thickened rather than cleared the fog of debate which envelops the colloidal lead treatment of cancer. It is not remarkable that this should be so, for the exact knowledge of the relation of a malignant growth to the organism bearing it is still lacking. The chorionic hypothesis of Beard and Blair Bell of the nature of cancer is far from satisfactory. The invasive and destructive appearances in the deciduate placentæ of rodents and primates is clearly much more due to the peculiar reaction of the maternal tissues in these orders than to primary properties of chorion (or trophoblast) as such. This may be inferred from the complete absence of these features in the non-deciduate placentæ (*e.g.* in the sheep), in which the villi dip into the uterine glands without entering into organic union with the maternal tissues. The conclusion is slowly gaining in cogency that the abortifacient action of lead as well as the ameliorations recorded in cancer cases are secondary to changes in the host tissues. From this point of view the lead treatment ranges itself with the various forms of radio-therapy, in which the clinical results are only intelligible as mainly consequent on an action on the supporting and nutritive stroma and not on a primary destructive action on the cancer cells themselves.

The investigations of Dr. Todd and his collaborators make a welcome objective addition to the elucidation of these intricate problems. Starting from the position taken up by Blair Bell in introducing the method, Dr. Todd has endeavoured to modify the treatment in the light of animal experiments and clinical experience in the direction of diminished toxicity of the colloid and greater uniformity in action. He brings a good deal of evidence to show that the uncontrollable toxicity of the lead-element colloids is due to their rapid passage to the ionised form, and has substituted colloidal preparations of

* "Chemotherapeutic Researches on Cancer," by A. T. Todd. M.D., M.R.C.P., Bristol; J. W. Arrowsmith Ltd., 1928. Pp. 127, Price 2s. 6d.

lead compounds, sulphide, selenide and telluride. Of these the selenide has proved the most satisfactory in his hands. In the animal experiments it is very significant that in a few animals (mice with implanted mammary carcinoma 63) recrudescence after apparently complete disappearance has occurred. This phenomenon, which is only too closely paralleled by some of the most successful human cases, is incompatible with the original hypothesis of a direct toxic action on the parenchyma cells, but quite intelligible if the action is indirect through the stroma. A remarkable and unexplained feature of the treatment of human cases is the striking analgesic action displayed. This has also been recorded for other colloidal preparations, and it would be of the greatest interest to know how it is brought about. The problem may be recommended to the physiologists as worthy of their attention.

Dr. Todd has given special prominence to the blood changes induced by these colloids. The effects in animals are employed as a preliminary test of toxicity, and in the clinical work repeated examinations of the blood are carried out to anticipate the development of the severe aplastic anæmia which is fatal to success. He has found that the administration of liver and liver extract permits of a higher dosage of colloid, and the accumulation of lead in the system is counteracted by the administration of potassium iodide.

Separate chapters are contributed on the preparation of the colloids and of the protective or stabilising substances.

What is the future outlook for the colloidal lead therapy in cancer ? If the views put forward in this review are sound, the method does not introduce a new chemotherapeutic principle. It achieves an alteration of the supporting and nutritive structures of the new growth of the same kind as that produced by X-rays and radium which may, in some cases, lead to regression or arrest of varying duration. As in so many other fields of therapeutics, *le mieux est l'ennemi du bon*, and until the possibility of a more rational direct chemotherapeutic attack is demonstrated we must surely encourage the development of any and every method which is capable of modifying the inexorable progressive march of malignant new growths.

Reviews of Books

A Short History of Medicine. By CHARLES SINGER, M.D., D.Litt., F.R.C.P. Pp. 368. Illustrated. Oxford: Clarendon Press. 1928. Price 7s. 6d.—This is a most interesting account of modern medical discoveries and successes, showing how they have followed during the last three or four centuries from the revived investigation of the laws of the physical sciences by measurement and experiment. It is prefaced by some all too brief chapters on the development of medicine from ancient times, especially on the Greek spirit and scientific method. One of the most striking chapters is the Epilogue, which discusses whether further progress is possible, or whether the engine which has carried us so far—"the scientific method," "the determinist thought,"—is not self-limited, although so many diseases are still not curable, and those men who reach a certain age have to-day no better chance of life than they would two hundred years ago. The story of the gradual growth of physiology, pathology and public health is beautifully told, though sadly limited by considerations of space and of the readers for whom it is designed. It may be said that this brilliant study of the principles underlying the science of medicine should not be called a history of medicine, but the author replies that it is a history of ideas in this branch of biological science, and the reader should bear this in mind.

How to Start in General Practice. By J. G. BRIGGS, M.R.C.S. Pp. 158. London: John Murray. 1928. Price 6s. —Hospitals do not equip men for private practice, it has been said lately, as pupilage prepared our forebears, and this is another book purporting to supply their insufficiency. The author has studied the means whereby, with little capital, place and foothold may be found, or a house economically constructed and adapted to medical needs. Indeed, he is rich in device. "Have a hidden push-bell from surgery desk to maid (who can be summoned to remind you of an appointment and so cut short the visit of a too loquacious

patient)!" In weakening the veracity of the maid the author lessens our interest in her master. Already, obviously, the latter is succeeding. One so wide awake and ready for emergencies, so handy with drugs, so thrifty, so methodical with records and accounts and so faithful to his own improvement, should not miscarry in his adventure nor fail in his lot.

Clinical Examination of the Lungs. By E. M. BROCKBANK, M.D., F.R.C.P. and A. RAMSBOTTOM, M.D., F.R.C.P. Second Edition. Pp. viii., 112. 35 illustrations. London: H. K. Lewis & Co. Ltd. 1928. Price 5s.—This small book should prove of real value to students and medical practitioners. In a short space it summarises clearly the theory and practice of the art of physical examination of the lungs in health and disease. Most of the statements with regard to physical examination are thoroughly sound and will pass without comment. Chapter IV., however, which deals with auscultation, serves to emphasise the deplorable lack of uniformity in nomenclature which makes the teaching of students so difficult at the present time. The classification of adventitious sounds and the use of the terms râle, crepitation and rhonchus by various authors differs so widely that one is forced, when speaking of another's description, to say that it doesn't much matter what he calls things if only he knows what he means. Chapter VII., which attempts to correlate physical signs with common pulmonary disease, is necessarily very sketchy, perhaps the book would be better without it. It sets out no new matter that is not already contained in the earlier chapters. The X-Ray plates at the end of the book are excellent.

Acute Aplastic Anæmia. By A. H. SMITH. Pp. viii., 80. 3 figures. London: H. K. Lewis & Co. Ltd. 1928. Price 6s. —This monograph forms a case report "par excellence." Clinical observations and methods of treatment are dealt with in full detail. The results of blood examinations provide numerous points of interest, and it is a remarkable feature of the case that the erythrocytes suffered little. A few observations on the normal blood picture in children would have been a useful addition to the section on differential diagnosis. The main feature, however, is the use of liver extract in treatment, and is of particular interest at the present time, especially as little success has yet been attained in aplastic anæmia.

Tuberculous Intoxications. By J. HOLLOS, M.D. Pp. x., 132. Edinburgh: E. and S. Livingstone. 1928. Price 10s. 6d. —As foretold in the preface, this book "will astonish many of us, it will shock others, it will convince some." The views expressed throughout this compilation of deductions by Dr. Hollos may seem to some to illustrate how a belief, perhaps brilliantly true at its conception, may so obsess the mind of a worker, that he sees in it the explanation of widely-different phenomena. That certain individuals may be hypersensitive to tuberculous toxins, and therefore display certain reactions, is no doubt true: but to claim, as does Dr. Hollos, that amongst these reactions are such widely-different manifestations as epistaxis and rheumatic arthritis seems a conclusion wholly unjustifiable. Dr. Hollos, from the nature of his research, perhaps knowingly and defiantly lays himself open to criticism, for he claims that only in those types of abnormality which do not present the usually accepted phenomena of tuberculosis, but which he assumes really to be of this nature, do these "lytic reactions" occur; an indulgent reader will perhaps tolerate these suggestions; but it requires an innocent credulity to accept the further conclusion that a few drops of immune blood, rubbed lightly into the skin, is the appropriate cure for dysmenorrhœa or a ganglion of the wrist. Unhappily, in the narrations of cases, we observe a vast degree of special pleading, and a zeal for victory, more than that which ought always to be the great aim of the investigator—the discovery of truth.

Fascial Grafting in Principle and Practice. By H. C. ORRIN, F.R.C.S. Pp. 92. 21 plates. Edinburgh: Oliver and Boyd. 1928. Price 7s. 6d.—The employment of fascial grafts and of living sutures has become very popular of late. Many articles advocating their use in a great variety of different conditions have appeared from time to time in various medical journals. Mr. Orrin has summarised the work of others besides embodying in this small volume the results of his own experience. He also includes some very useful technical hints. Most surgeons have probably employed fascial grafting for the cure of ventral and recurrent inguinal hernias. To many, however, its use as a means of remedying adherent scars, for defects of the bladder, urethra, ureter, for fæcal fistula, or to improve the condition following a facial paralysis or ptosis, might not suggest itself. The book is a short one and easily read. It contains good photographs, a useful bibliography and a large number of split infinitives.

Handbook of Medical Jurisprudence and Toxicology. By W. A. BREND, M.A., M.D., B.Sc., Barrister-at-Law. Sixth Edition. Pp. xiv., 327. London: Charles Griffin & Co., Ltd. 1928. Price 10s. 6d.—At the present time when a medical curriculum is crowded with an increasing number of subjects, it is essential for teachers and students alike to make some distinction regarding the relative importance of the varied objects of study. It is obviously impossible for anyone to become an expert medical jurisconsult or a toxicologist without long and serious study, but it is not difficult to obtain a working knowledge of forensic medicine and toxicology in a short time by a judicious use of text-books. This selection is largely in the students' hands, and we are acquainted with no work which deals with the subjects referred to in a clearer way than does this small volume written by Dr. Brend. The author has spared no pains to render the work suitable for use by medical students and practitioners, it is in no sense a "cram book," but is written in such a lucid way that the reader imbibes accurate knowledge with little effort. The various sections are well up to date—as evidenced by the inclusion of recent tests for blood and references to new statutory enactments affecting medical practitioners. The convenient size and clear print contribute in no small degree to the excellence of the book, and we have no hesitation in recommending it to all those who wish to obtain a working knowledge of a very interesting subject.

Clinical Surgery. By J. W. DOWDEN, M.B., F.R.C.S. Pp. 68. Edinburgh: Oliver and Boyd. 1928. Price 2s.—The issue of this small volume comes as a welcome addition to the numerous text-books which a medical student is called upon to read. This is saying a good deal, nevertheless it is true. Of the sixty-eight pages there is not a dry one. The book is written in Mr. Dowden's pleasing style, and deals with the management of any surgical case from first to last. A few general hints to the beginner precede a chapter on facts which may be learnt by cultivating one's power of observation. Then follows some sound advice on the systemic examination of a patient and a guide to history taking. A few pages are devoted to certain principles of treatment, and a brief paragraph deals with prognosis. The order of case taking advised is not that universally accepted, but this is a minor point. Every student would do well to read and almost to learn the book by heart, before actually commencing hospital appointments. Such a proceeding would be of inestimable benefit to many students and patients.

A Code of Rules for the Prevention of Communicable Diseases in Schools. By THE MEDICAL OFFICERS OF SCHOOLS ASSOCIATION. Ninth Edition. Pp. 74. London: J. and A. Churchill. 1928. Price 2s. 6d.—The most important alterations in this edition are the more precise and detailed directions in reference to chemical disinfection, and a most excellent appendix summarising the present position in regard to the recognition of susceptibility to diphtheria and scarlet fever, and of the methods of immunisation and treatment by means of their several antitoxins. This code of rules, coming from so authoritative a source, will do much to bring about uniformity of practice in public and preparatory schools. The information provided will prove of the greatest service, not only to School Medical Officers, but to general practitioners, and all who are responsible for the health of children at school.

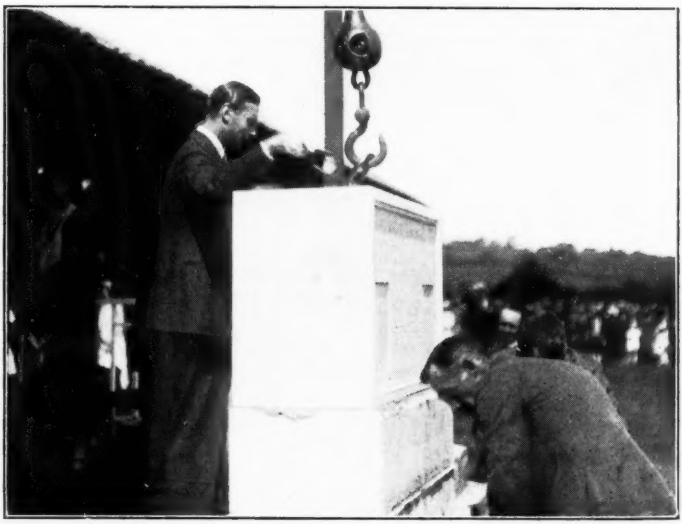
Diseases of the Larynx. By HAROLD BARWELL, F.R.C.S. Third edition. Pp. xvi., 278. Illustrated. London: Oxford University Press. 1928. Price 12s. 6d.—The third edition of this excellent little manual follows at rather a long interval after the second. The eighteen intervening years have witnessed so great a development that it has been necessary to rewrite the whole book. Nevertheless, the size has not materially increased, in spite of generous print and numerous and handsome illustrations: the latter have the great merit of being drawn by the distinguished laryngologist-author. A volume devoted to diseases of the larynx and œsophagus only, and intended for the student and practitioner rather than the specialist, is not often seen nowadays, but those in search of such a work will not find a better.

Crawford W. Long and the Discovery of Ether Anæsthesia. By F. L. TAYLOR, M.D. Pp. xiv., 237. 8 plates. London: Oxford University Press. 1928. Price 18s.—Mrs. Frances Long Taylor wrote this account of her father, Dr. Long's life, in order that his direct descendants might have a record of his exceptional career and valuable work. The book was intended for circulation among members of the Long family, and for this reason contains much domestic detail; but it affords us a very vivid picture of a progressive man making good in spite of adverse circumstances. We read that Dr. Long was convinced, by *post-mortem* evidence, that pulmonary tuberculosis was a curable affection, and as a result he treated phthisical patients with open air and special dieting. In surgery his technique as regards carcinoma was evidently well in advance of that of his time, for he took infinite pains

when operating upon a breast to clear out axillary glands and remove soft tissues from the ribs. On the outbreak of the American Civil War, one of his first precautions was to lay in a large store of iodine for the treatment of wounds. His most important discovery occurred in 1842, when he made the first recorded use of ether as an anæsthetic during a surgical operation. In this connection his fame has now become world wide, and the justice of his claim to priority in the employment of ether anæsthesia has been established by the evidence brought forward by Mrs. Taylor, a fact which makes this book a valuable addition to the archives of medical evolution.

The Medical Annual, 1928. Forty-sixth year. Pp. c., 630. Illustrated. Bristol: John Wright & Sons. 1928. Price 20s. —We think that the publishers and editors have every reason to be pleased with this year's issue, for the reader will find that it contains a specially rich harvest of medical and surgical gains, many of them no doubt of minor importance, though some bid fair to be of great and permanent value. Thus the discovery of a serum which aborts poliomyelitis will be of immense value if the supply can be largely increased and really early diagnosis ensured. Possibly the latter difficulty may be met if some test can be found like the Schick reaction for diphtheria. We should like here to draw attention to the beautiful coloured plates which are given of this test, and of the scarlatina one, with the account of the results obtained. The present volume, indeed, has no less than 53 plates and 130 illustrations, some of them of high artistic merit. Among the many attractive articles we may instance the account of the liver treatment of pernicious anæmia and a useful discussion of our present knowledge of the disease; a similar discussion on epilepsy, describing the ketogenic diet for young patients which seems to have given some cures; a practical article on sprains and synovitis; one on the great help given by anti-gas serum in abdominal operations, and not least, one on the management of boarding schools during epidemics with its many trying problems. There are, too, records of the discovery of an organism peculiar to trachoma; of an antidote for cocaine poisoning; a new treatment for burns by tannic acid; and one for drug addiction by narcosan, a solution of lipoids, which gave striking results when used in New York on 366 patients. Readers will highly appreciate the handy directories and lists of new books, instruments and drugs which are an important feature of this most necessary book.

PLATE VI.



ROYAL VISIT TO WINFORD.
JULY 7TH, 1928.

Editorial Note.

The Royal Visit to Winford.

On July 7th the Duke and Duchess of York came to Bristol to lay the foundation stone of the new open-air hospital at Winford. The weather was beautifully fine, and—thanks to the excellent arrangements made by those in charge—a great many interested citizens were enabled to watch the ceremony and to see for themselves the beauty of the surroundings in which the new hospital is to be built. The purposes of this hospital were explained by Miss F. M. Townsend, J.P., Chairman of the Bristol Crippled Children's Society, in a little speech which was a model of compactness and lucidity. One point on which she laid particular emphasis deserves notice, and that is the example of union which this new movement sets to the other institutions of Bristol. The Bristol Crippled Children's Society and the Bristol Orthopædic Hospital have found it possible to sink their differences and pool their assets, and this ought to prove the first instalment of a larger and more general process of hospital union such as is taking place in most of the University towns of this country.

In this connection it should be noted that the site purchased is larger by far than the present needs of the open-air hospital demand. About seventy acres are available for the building of country annexes to the

other voluntary hospitals of the city, if and when these are thought necessary. This site was chosen most carefully, geological and other experts being consulted, and it was in the belief that it was the best, from every point of view, that the neighbourhood of Bristol could furnish that it was chosen. (In passing it may be noted that the upper of the two pictures which we publish shows Sir Robert Jones applauding vigorously. This is by no means the only expression of his approval of the site. He visited it early in the negotiations for purchase, and was unequivocal in his praise.) Before the site was bought the principal officers of the chief voluntary hospitals of the city were consulted, and though they were not in a position to commit their institutions actively to the support of the movement, it was understood that they did not, in principle, disapprove of it. Before long it is probable that some one of these institutions will give a lead to the others by planning an extension of its accommodation in the form of a country annexe on the Winford site.

A third point of importance on which Miss Townsend dwelt was the fact that this country hospital embodies a form of union between voluntary and State effort which may prove to be a valuable precedent. The building of the hospital is in the hands of a voluntary committee, while the income which pays for its upkeep will be largely derived from Board of Education grants. It seems possible that this characteristically British plan of compromise may preserve for the hospital movement that freshness and originality which marked the voluntary era, while adding to this the stability that is ensured by State support.

Finally, Miss Townsend found it necessary to emphasise the fact that this movement needs a great deal of money. The present position is this, that the

cost of the undertakings already entered upon is approximately £50,000. Towards this a sum of over £25,000 has been subscribed or promised. The appeal for subscriptions to meet this deficit will, we feel sure, be all the more successful because it is made on behalf of a movement that initiates a policy of hospital union in Bristol.

The Right Hon. the Viscount Haldane, K.T., O.M.

WE record with great sorrow the death of the Chancellor of the University of Bristol, LORD HALDANE, on August 19th, 1928. Our Chancellor was more than a titular head of the University: he took a keen and active interest in its progress and paid frequent visits to Bristol, his last being on the occasion of the Degree Congregation in June of this year. Of his eminence as a statesman and a lawyer this is not the place to speak. But a medical journal may at any rate express appreciation of him as a philosopher, and pay some meed of tribute to a great War Minister and patriot.

Meetings of Societies.

Bristol Medico-Chirurgical Society.

February 8th, 1928.

DR. W. K. WILLS, President, in the Chair.

DR. S. E. DORE read a paper on "**Radiation Therapy in Dermatology,**" reported on page 85 of our last issue.

March 14th, 1928.

DR. W. K. WILLS, President, in the Chair.

DR. A. T. TODD gave a further account of his "**Chemotherapeutic Researches on Cancer,**"[1] an appreciation of which appears on page 217.

This was preceded by a short address on the "**Colloidal State of Matter,**" by Professor Francis. The report of this address, which was illustrated by many experiments, appears on page 177.

In the discussion which followed Dr. Todd's paper, DR. R. S. STATHAM spoke of fourteen cases of malignant disease of the pelvic organs treated with lead. In two instances he had observed great improvement. In some of those that had not been so fortunate cicatrisation following arrest of the growth had brought about ureteral obstruction. The relief from pain had been remarkable.

DR. D. A. ALEXANDER described the regression of a breast tumour under the influence of treatment. The patient had ultimately died of metastases.

PROFESSOR D. C. RAYNER corroborated Dr. Statham's account of the favourable reaction of an ovarian carcinoma.

MR. C. FERRIER WALTERS spoke of the help afforded by lead in the treatment of two malignant growths of the prostate, surgically removed.

April 11th, 1928.

DR. A. L. FLEMMING in the Chair.

MR. A. G. TIMBRELL FISHER, M.C., read a paper on "**Manipulative Surgery,**" part of which is reported on page 165.

[1] Bristol: J. W. Arrowsmith Ltd. 2s. 6d.

May 9th, 1928.

DR. W. K. WILLS, President, in the Chair.

DR. G. B. BUSH showed a group of radiograms illustrating bone pathology.

MR. G. W. ROSS showed a case of abdominal swelling, possibly hydatid disease. MR. C. A. MOORE mentioned that he had recently had a case of hydatid disease from Wales and asked what was proposed. The PRESIDENT suggested X-ray treatment on the assumption that this was a case of Hodgkin's disease.

MR. E. WATSON-WILLIAMS showed four cases of cancer of the tonsil in elderly men treated by selenium, with and without radium in addition. He emphasised the difficulties which arose from patients presenting themselves for treatment only when the disease was advanced, with enlargement of glands.

DR. P. WATSON-WILLIAMS discussed this point and the technique of treatment in inoperable cases.

DR. HERBERT ROGERS read a paper on **"The Estimation of Chlorides in the Cerebro-spinal Fluid,"** reported on page 197

The Bath and Bristol Branch of the British Medical Association.

May 30th, 1928.

AT a Clinical Meeting, held on Wednesday, May 30th, at the University, MR. A. W. ADAMS showed a series of cases of club-foot, and cases of malignant disease of the alimentary tract.

DR. R. C. CLARKE showed a series of cases and pathological specimens of congenital heart disease.

MR. H. CHITTY showed cases of cervical rib and of congenital deformity.

MR. E. W. HEY GROVES showed cases illustrating the results obtained by operation in the treatment of injuries to limbs.

MR. C. A. MOORE showed a case of sciatic tumour.

DR. PERCY PHILLIPS showed cases of syringomyelia.

DR. A. T. TODD showed cases of amyotrophic lateral sclerosis and others.

MR. E. WATSON-WILLIAMS showed cases of atresia and hyperostosis of the external ear and others illustrating results obtained by operative treatment of malignant disease of the larynx.

DR. KENNETH WILLS showed cases of rodent ulcer and other dermatological diseases.

DR. A. D. FRASER showed pathological specimens.

The Medical Library of the University of Bristol,

WITH WHICH IS INCORPORATED

The Library of the Bristol Medico-Chirurgical Society.

The following donations have been received since the publication of the List in June, 1928.

	September, 1928.
Liège Université Bibliothèque (1)	1 volume
Michell Clarke Memorial Fund (2)	1 ,,
Dr. H. B. Murgoci (3)	1 ,,
New York University and Bellevue Hospital Medical College (4)	1 ,,
Mr. W. M. Ogilvie (5)	1 ,,
Rockefeller Foundation (6)	1 ,,
Royal College of Physicians of London (7) ..	1 ,,
Sheffield University, Medical Faculty (8) ..	1 ,,
Surgeon-General of the United States Army (9)	1 ,,

Unbound periodicals have also been received from Professor E. W Hey Groves and Dr. Carey Coombs.

THE ONE HUNDRED AND TWENTY-FIFTH LIST OF BOOKS.

The figures in round brackets refer to the figures after the names of the donors. The books to which no such figures are attached have either been bought from the Library Fund or received through the *Journal*.

Brend, W. A.	*Handbook of Medical Jurisprudence* .. 6th Ed.		1928
Gravis, A.	*Récherches anatomiques et physiologiques sur le tradescantia virginica L.*	(1)	1898
Medical Department of the United States Army in the World War	Vol. 9	(9)	1928
Notes on Clinical Laboratory Methods		(3)	1927
Ogilvie, W. H.	*Recent Advances in Surgery*	(5)	1928
Orrin, H. C.	*Fascial Grafting in Principle and Practice* ..		1928
Osler and McCrae ..	*Modern Medicine.* Vol. 6 .. .. 3rd Ed.	(2)	1928
Porter, W. S.	*The Medical School in Sheffield*, 1828-1928	(8)	1928
Tercentenary Celebration of the Publication of the "De Motu Cordis" by William Harvey, 1628.	*Programme of Arrangements*	(7)	1928
Todd, A. T...	*Chemotherapeutic Researches on Cancer*		1928

TRANSACTIONS, REPORTS, JOURNALS, ETC.

Collected Reprints from the Department of Experimental Surgery, New York University and Bellevue Hospital Medical College. Vol. 5 .. (4) 1926-27
Methods and Problems of Medical Education. 9th Series .. .. .. (6) 1928
Progressive Medicine, 1928. Vols. 1 and 2 1928
Surgical Clinics of North America. Vol. 8. No. 3 1928

Local Medical Notes.

Dr. MacDonald Critchley has been appointed Assistant-Physician to the National Hospital, Queen Square.

EXAMINATION RESULTS.

University of Bristol.—Students of the University have recently passed the following examinations :—

M.B., CH.B.—*First Examination. Pass :* S. B. Adams, A. J. Board, G. M. Evans, Miss M. A. W. Hawkes, F. J. W. Lewis, R. L. Marks, H. E. Pearse, Miss F. E. Powell, S. L. Prescott, O. E. L. Sampson, J. P. P. Stock.

M.B., CH.B.—*Second Examination.* (*Part I.*). *Pass :* G. M. Evans, F. E. Fletcher, R. L. Marks, J. P. P. Stock. *In Organic Chemistry* (*Completing Examination*) *:* J. Ridgway. *In Elementary Anatomy* (*Completing Examination*) *:* G. T. Bevir, W. R. Cambridge. *In Elementary Anatomy only :* H. James.

M.B., CH.B.—*Second Examination.* (*Part II.*). *Pass :* J. S. Adamson, J. E. Brown, C. J. N. Davis, L. C. Holland, C. N. Royal.

FINAL M.B., CH.B. (*Part II.*).—*Completing Examination. Pass with 2nd Class Honours :* T. H. Berrill (with distinction in Special Pathology). *Pass :* B. J. Boulton, R. D. Jenkins (with distinction in Public Health), Miss H. B. Murgoci (with distinction in Special Pathology), T. B. Wansbrough (with distinction in Obstetrics). *In Group II.* (*Completing Examination*) *:* D. E. C. Andrew, A. J. McD. Grimston.

B.D.S.—*Second Examination. Pass in Physiology and Anatomy* (*Completing Examination*) *:* M. J. Ryan.

FINAL B.D.S.—*Pass :* W. M. Evans.

L.D.S. (*Preliminary Science*).—*Pass : In Biology only* (*Completing Examination*) *:* Miss H. C. L. Blinkworth, R. E. W. Smith.

L.D.S.—*First Professional Examination. In Physiology and Anatomy (Completing Examination). Pass:* E. V. O'Hara, H. L. Saunders. *In Physiology and Anatomy only. Pass:* A. MacDonald Watson.

L.D.S.—*Second Professional Examination. In Dental Mechanics and Metallurgy (Completing Examination). Pass:* A. N. Moon, T. A. Satchwell, C. G. Westlake. *In Materia Medica (Completing Examination). Pass:* Miss B. J. Shapland. *In Dental Metallurgy and Mechanics only. Pass:* J. D. Galloway. *In Materia Medica only. Pass:* E. R. Carpenter. *In Dental Metallurgy and Materia Medica only. Pass:* I. W. Williams.

L.D.S.—*Final Professional Examination. Pass:* J. H. Edwards, H. J. Phillips, Miss S. M. Weir.

CONJOINT BOARD.—M.R.C.S., L.R.C.P.—*Preliminary Medical:* R. H. Moodie, C. A. G. Evans.

M.R.C.S., L.R.C.P.—*First Examination. Anatomy and Physiology:* D. P. Dewe. *Physiology:* Miss A. M. B. Walker. *Pharmacy and Materia Medica:* C. H. Pauli.

M.R.C.S., L.R.C.P.—*Final Examination:* H. D. Pyke (*Medicine and Surgery only*); A. C. Fisher (*Medicine, Surgery and Midwifery, Completing Examination*); L. E. Vine (*Medicine, Surgery and Midwifery, Completing Examination*); L. H. Wharton (*Midwifery only*); D. R. House (*Surgery and Midwifery, Completing Examination*); T. H. Berrill (*Surgery and Midwifery, Completing Examination*); Miss C. F. Fox (*Surgery, Completing Examination*).

M.R.C.S., L.R.C.P.—*Diploma in Tropical Medicine and Hygiene:* E. R. Wide (M.B., Ch.B., Bristol).

L.D.S., R.C.S.—*Final Examination (Part I.):* H. J. Phillips, R. G. Boodle, C. Haskins.

L.D.S., R.C.S.—*Final Examination (Part II.):* H. J. Phillips, R. A. Phillips.

SOCIETY OF APOTHECARIES.—L.S.A.—*Forensic Medicine:* T. A. Barnabas.

L.S.A.—*Midwifery:* T. A. Barnabas, N. H. Kettlewell, L. P. Gregory.

L.S.A.—*Medicine:* T. A. Barnabas.